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UNITED STATES DISTRICT COURT
DISTRICT OF OREGON
EUGENE DIVISION

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| MCKENZIE FLYFISHERS, STEAMBOATERS,<br><br>        Plaintiffs,<br><br>        vs.<br><br>BRUCE MCINTOSH, SCOTT PATTERSON, OREGON DEPARTMENT OF FISH AND WILDLIFE,<br><br>        Defendants. | Case No.: 6:13-cv-02125-TC<br><br>**DECLARATION OF DR. GORDON LUIKART IN SUPPORT OF PLAINTIFFS' MOTION FOR SUMMARY JUDGMENT AND INJUNCTIVE RELIEF** |

I, Gordon Luikart, hereby declare:

1.      I am a Professor of Fish and Wildlife Genetics at the Flathead Lake Biological Station, University of Montana. My address is 41229 Haystack Mountain Lane, Polson, Montana, 59860. I have nearly 20 years of professional experience researching and teaching at the university level. Since 2000, I have specialized in research and teaching in the area of animal conservation, ecology, and population genetics/genomics.

2.      From 1997-2000, I was a postdoctoral research scientist in Europe working on large (five country) animal genetics projects funded by the European Union and also the National Science Foundation and the North Atlantic Treaty Organization. I received a Ph.D. in Organismal Biology and Ecology in 1997 and a M.S. in Zoology in 1992, both from the University of Montana (including a year at two Australian universities on a Fulbright fellowship). I received a Bachelor of Science degree in Biology from Iowa State University in 1988, with a minor in Animal Ecology.

3.      Since 2005, I have spent part of my years researching and teaching as a visiting professor and senior research scientist at the Center for Investigation of Biodiversity and Genetic Resources at the University of Porto in Portugal. For the last four years, I have spent the majority of each year researching and teaching as a professor at the University of Montana's Flathead Lake Biological Station. I teach courses including advanced graduate level and undergraduate level conservation ecology, conservation genetics, and fish and wildlife genetics/genomics (population genetic data analysis; http://www.popgen.net/congen2013/).

4.      I have conducted genetic research on fish and wildlife populations in several different countries in addition to the United States and Portugal, including in Australia as a Fulbright Fellow, and in France as a government scientist, where I won the bronze medal as a top researcher with the Centre National de la Recherche Scientifique (CNRS). I have researched a wide range of species, including goats and other ungulates, carnivores, fish, and other aquatic species. My research and teaching embraces various topics related to animal genetics/genomics, including disease diagnostics (via pathogen DNA testing), population genetics theory and modeling, adaptation in captive and domesticated populations, endangered and threatened

species recovery, effects of gene flow and hybridization (introgression) on individual fitness, population size and structure, local adaptation in natural environment, and monitoring of the genetically-effective population size and the effective number of breeders using genetic markers in natural populations.

5.　　I have conducted numerous research investigations on these subjects. I have served as a principal or co-principal investigator on more than 40 scientific research projects, and my work has produced chapters in four books, all relating to animal population genetics (one on animal domestication), and over 100 scientific papers in peer-reviewed international journals. In 2007, I co-authored a major textbook on conservation genetics, the second edition of which was published in 2013, and contains updated sections relevant to salmon hatcheries, introgression, and their effects on effective population size and fitness in wild stocks.

6.　　My recent research projects involved developing a computer program simulator to model landscape level gene flow for aquatic species in complex river systems, and peer-reviewed publications on DNA sequencing and gene flow in trout and salmon, and in wild and domestic populations (including development of novel estimators of the number of breeders (spawners) per generation from genetic marker data). My scholarship includes service on the editorial board of the journal *Conservation Biology* and as an associate editor for both *Molecular Ecology Resources* and the *Journal of Heredity*. In 2014, I was recognized as "one of the world's most influential scientific minds" by Thomson Reuters, for my research and publications over the past decade (see report at http://sciencewatch.com/sites/sw/files/sw-article/media/worlds-most-influential-scientific-minds-2014.pdf.).

7.　　In the last five years, I was recruited as an advisor for the Swan Ecosystem Center Native Fish Committee. I have been a member of the American Fisheries Society, the Ecological Society of America, the Society for Conservation Biology, the Society for the Study of Evolution, the Wildlife Disease Association, and the Wildlife Society. Attached hereto as Exhibit A is my curriculum vitae, which include a list of peer-reviewed publications I have authored since 1996. I have substantial familiarity with the genetics of animal domestication, adaptation to captivity (i.e., domestication), the effects of gene flow and introgression on fitness and

population persistence in fish and wildlife, local adaptation in salmonids, and statistical and molecular empirical genomics.

## Summary of Opinions.

8.     The McKenzie Hatchery spring Chinook salmon program significantly harms wild spring Chinook salmon in the McKenzie River basin and significantly reduces the ability of the wild spring Chinook salmon population to recover. Hatchery-bred salmon and their offspring likely have substantially reduced fitness compared to wild salmon due to decades of strong domestication selection for adaptation to captivity and the hatchery environment. Adult hatchery-bred Chinook cross-breed with wild Chinook, and it is likely that the resultant offspring have reduced fitness (i.e., survival and reproduction) in the wild. Some maladapted genes introduced by hatchery-bred Chinook into the wild Chinook population could persist for many generations in the wild population. This harm is especially problematic and worrisome in the context of the McKenzie River basin, because the McKenzie River wild Chinook population is a "genetic legacy" population, critical to recovery of the Upper Willamette River Basin ESU. The McKenzie Hatchery spring Chinook program should be eliminated or greatly reduced so that it collects far fewer wild spawners for broodstock (<30 individuals, including mainly or only males), and avoids the harm that results from the genetic effects of large-scale hatchery smolt releases. Harm caused by hatchery smolt releases should be limited (or eliminated) by releasing fewer hatchery smolts into the wild in the basin (≤77,000; assuming a return rate to the hatchery of ~0.59% and the HSRG's baseline of 5% pHOS), which should allow the wild Chinook population to begin to recover its productive capacity in the context of current and future environmental selective regimes. Finally, more powerful genomic monitoring is crucially needed for all McKenzie River spring Chinook to accurately quantify the amount of introgression from hatchery fish into wild populations (including introgression of maladaptive genes) in the basin, and to quantify the effects of this introgression on the fitness (reproductive success, survival and growth) of the wild fish, in the wild.

## The Genetic Importance of Wild Spring Chinook in the McKenzie River Basin.

9.      Spring Chinook salmon in the McKenzie River basin comprise the sole "genetic legacy" population in the Upper Willamette River basin, and are designated a "primary" population, meaning they are "of the highest biological significance" among spring Chinook salmon in the Upper Willamette River ESU. As a geneticist, these determinations mean to me that the McKenzie River wild Chinook population is the most important population within the ESU to conserve in order to maintain the adaptive potential and ensure the persistence of the entire ESU. This includes conserving the genetic integrity (wild ancestry) of McKenzie River wild Chinook population.

10.     These determinations should be put in the context not only of the Upper Willamette River ESU, but also in context of the habitat for, and status of, wild salmonids in the lower 48 states. Studies show that by building dams we have eliminated nearly one-half of once-accessible habitat for wild salmonids in the Pacific states. Because of dams and other factors, in the Pacific Northwest alone, some 37 genetically distinct salmon runs have been lost forever, including wild coho salmon in the Snake, Grande Ronde, Yakima, Walla Walla, and Bull Run Rivers; wild sockeye salmon in the Metolius and Wallowa Rivers; wild fall Chinook salmon in the Willamette and Umatilla Rivers; and wild spring Chinook salmon in the Lewis, White Salmon, and Klickitat Rivers. In my view, any remaining genetically distinct or viable population of wild salmon or steelhead trout is indispensable not just in the context of any ESU or DPS in which it is listed, but in the broader context of the species' range as a whole.

**Broodstock Used in the McKenzie Hatchery.**

11.     The February, 2014 proposed HGMP for the McKenzie Hatchery states at 6.2.1. that the broodstock in current use ("Stock 23") is derived primarily from adult returns to the McKenzie River basin. The February, 2014 HGMP states that since 1990, broodstock for the hatchery program has been derived entirely from Chinook salmon collected at McKenzie Hatchery and occasionally at Leaburg Dam on the McKenzie River. However, the earlier history of Stock 23 is mixed and less certain. The February, 2014 HGMP notes that other Willamette River stocks have been incorporated into the broodstock at the McKenzie Hatchery over the years, and that there is evidence that strays from other hatcheries were incorporated into the broodstock. The record in

this case includes ODFW documents, some with unknown authors and dates, that incompletely describe a series of out-of-basin, in-and-out transfers on many occasions during the lengthy period of 1908-1993. In summary, the McKenzie Hatchery fish genetic origins are apparently diverse, uncertain, but mainly from the adult returns to the McKenzie River basin. This is a critical issue as these uncertainties as to origin make the impact of interactions between hatchery and wild origin fish highly questionable, and likely more damaging to the genetics of the McKenzie wild stock (e.g., reducing fitness and adaptation in the wild fish more than if hatchery origins were 100% from the McKenzie).

12.      The October, 2014 HGMP states that the McKenzie Hatchery is ostensibly an "integrated" program, meaning it is intended to use both wild and hatchery spring Chinook in the broodstock, with a current goal of collecting 600 fish for broodstock. The October, 2014 HGMP states that the hatchery has a goal of integrating 5%-10% wild Chinook into the broodstock annually. Between 2002 and 2012, a range of 1.2% to 10.1% (with an average of 4.1%) of the hatchery broodstock were reported to be wild (non-fin-clipped) spring Chinook. More recent analyses of salmon returning to the hatchery show that up to 80% of the fish previously assumed to be wild, and incorporated into the broodstock, were actually mismarked hatchery fish. The true level of incorporation of wild Chinook into the hatchery broodstock would thus be lower (potentially substantially lower) than originally reported. The true level of incorporation makes the McKenzie Hatchery somewhat similar to a "segregated" hatchery, or intermediate to a segregated and integrated hatchery, which is the most problematic kind of hatchery, because it can lead to or cause the highest reductions of fitness in the wild (Baskett and Waples 2013). The Hatchery Scientific Review Group (HSRG) has stated that "intermediate" hatcheries should not exist at all (see paragraphs 34 and 35, because they provide neither the possible benefit of hatchery and wild fish separation that is central to segregated hatcheries, nor the possible benefit of continuous and sufficient genetic infusion from the local wild population that is central to integrated hatcheries.

13.      The Oregon Department of Fish and Wildlife (ODFW) asserts that currently it does not include wild spring Chinook in the broodstock in the McKenzie Hatchery, but plans to reinitiate

wild broodstock incorporation in the future at a level of 5-10%. In his rebuttal disclosures submitted during discovery in this case, Dr. Marc Johnson states that "inclusion of wild fish into the McKenzie hatchery <u>appears</u> to meet its intended goal" (emphasis added) of providing an integrated hatchery broodstock. Unfortunately, it cannot now be known if the goal is met, without genetic monitoring data from many informative DNA markers, far more than have thus far been used by ODFW (see below). This is required because there is "low genetic differentiation" at some/most genome locations (loci) genome-wide, except at important genome regions likely selected for adaptation to captivity (domestication). Further, the true rate of infusion of wild genes cannot now be known because a substantial proportion of the unclipped individuals are (1) known to actually be of direct hatchery origin because 5% of hatchery released fish are not clipped (hatchery-marked), (2) a proportion of marked fish regenerate (regrow) their clipped fin (obscuring the 'hatchery mark'), and (3) many so-called wild (natural) fish actually have hatchery ancestry (parents or grandparents) so they are not contributing only wild genes needed for infusing in an integrated hatchery.

14.     In his rebuttal disclosures during discovery in this case, Dr. Johnson questions the appropriateness of describing the McKenzie Hatchery population as "intermediate." However, it is clear that the McKenzie Hatchery population is best characterized as an "intermediate" hatchery for four reasons, even if mismarked fish (not fin clipped) are accounted for in the estimated rates of integration of wild fish, for the following four reasons:

(1)  The PNI (proportionate natural influence) is (far) below the 0.67 threshold which is the HSRG guideline for a "primary population" like the McKenzie Chinook salmon. (See HGMP for values for pHOS and pNOB, which are the components of PNI. The recent average pHOS is listed as 39%; and pNOB has been in the 1.2-10.2% range, but in 2013 was zero). Further, the guideline states that in all cases the pHOS for an integrated hatchery must be less than 30%, which according to ODFW annual reports has not been achieved on the McKenzie for many years. The HSRG guidelines (HSRG 2009, White Paper No. 1.) for integrated and segregated hatcheries are the only standards that are

widely recognized to have a well documented, published scientific basis. Dr. Johnson does not provide or cite an alternative authoritative standard for an integrated hatchery.

(2) As mentioned above, a substantial proportion of 'natural origin' fish have considerable hatchery ancestry (which likely makes the actual rate of natural origin infusion much lower than 4.1%).

(3) The current interim period (2012 to HGMP approval date) provides NO wild fish integration (allowing for rapid adaptation to captivity).

(4) The run (return) timing is substantially different (later) for the McKenzie Hatchery fish compared to wild fish (recently confirmed in deposition; Sharpe Dep 24:19), suggesting adaptive (maladaptive) divergence between the hatchery and wild populations. The difference in peak run time for hatchery fish and for natural origin fish suggests hatchery differentiation and adaptation to captivity.

Similarly, in his deposition, Cameron Sharpe testified that the McKenzie Hatchery is not integrated but "is currently segregated."

**The Johnson and Friesen Study.**

15.     The February, 2014 HGMP at 6.2.4. states: "Using genotypic data for 13 microsatellite markers, Johnson and Friesen (2012) found no significant genetic difference between hatchery and natural-origin spring Chinook sampled in 2011 from the McKenzie River (H$_0$: θ = 0; p > 0.05)." Similarly, the document *Reintroduction Planning: Management of Spring Chinook Salmon above Cougar Dam South Fork Mckenzie River 2013 (December 2012 draft)*, cites the Johnson and Friesen study, and states: "[T]here may be domestication genetic risks of using hatchery fish." McKenzie hatchery and natural spring Chinook salmon are very similar genetically (Figure 3); so the risk of using hatchery fish for reintroduction should be very low in terms of impacting the genome."

16.     Neither of these broad presumptions (of no genetic differentiation, and very low risk of impacting the genome) is supportable. Johnson and Friesen "used multilocus microsatellite genotype data from 813 spring Chinook salmon *Oncorhynchus tshawytscha* to investigate patterns of genetic diversity within and among wild and hatchery populations from the

Willamette River. An analysis of genetic divergence (θ) revealed little or no differentiation between hatchery and local wild populations within Willamette River subbasins." However, the authors state: "We emphasize that these results reflect changes predicted for neutral microsatellite loci, and simulations did not incorporate potential effects from selection on allele frequencies and overall population genetic diversity." The authors caution: "We again emphasize that the models of genetic change we have presented represent dynamics that might be expected for neutral loci. Allele frequencies for genes subject to selection can be expected to respond not only to demographic variables such as migration and population size, but also to the direction and magnitude of selection. Though we found no evidence for selection having influenced GAPS or *TKU* microsatellite allele frequencies among Willamette River spring Chinook populations, *it is possible that other genetic loci, not considered in this study, could be subject to disruptive natural or artificial selection.*" (emphasis added)

17.     It is fundamentally improper to use solely microsatellite (DNA) allele frequencies to determine whether significant adaptive differences exist between hatchery and wild fish, because the genetic structures that are assayed (only very few non-functional genome segments) are not generally relevant to the important adaptations of the stocks in question. My conclusion in this respect is fully supported by the Independent Scientific Review Panel (ISRP) report 2011-26, at 12 (Dec. 2011), which states: "In one of the FY 2012 proposals (AP-6 spring Chinook genetic diversity), microsatellite allele frequencies in hatchery and wild fish were going to be used to infer whether hatchery management was preserving life-history variation. That is, if the hatchery stocks contained the variation extant in the wild fish and allele frequencies were similar, then the implication is that genetic attributes responsible for life history variation were being preserved. This argument is not supported by empirical or theoretical justification. Allele frequency variation could be used to evaluate genetic drift, inbreeding, effective population size, and such. *But important selection could be taking place at loci not being tracked by the neutral loci.*" (emphasis added.)

18.     Similarly, as far back as 1995, the National Marine Fisheries Service (NMFS) convened a panel of experts to consider effects of genetic introgression in salmonids. The experts expressed

similar concerns about genetic interpretation of allele frequencies, finding: "More modest genetic differences may not result in such large, short-term reductions in productivity, but persistent gene flow would probably cause the replacement of local genes with non-native ones. *Genetic distances derived from molecular genetic data may not reflect adaptive differences between hatchery and natural populations.*" (NMFS 1997. NOAA Tech Memo NMFS-NWFSC-30: Genetic Effects of Straying of Non-Native Hatchery Fish into Natural Populations. Proceedings of the Workshop June 1-2, 1995). (emphasis added)

19.     Johnson and Friesen found no evidence for positive selection on four immune-relevant loci (Tonteri et al. 2008), as allele frequencies at these markers were very similar for the hatchery-bred and wild Chinook populations in the McKenzie River basin. It is not surprising that there is genetic *similarity* between the hatchery-bred and wild spring Chinook populations, given that: 1) the hatchery brood stock was founded by local, spawners; 2) wild fish have been integrated into the hatchery brood stock and; 3) a proportion of hatchery-origin fish spawn in the wild. Since it is conceded that the there is no reason to believe that the microsatellite DNA loci are subject to natural selection (and that so few loci were used), the results of this study can tell us little regarding the similarities between the wild and hatchery stocks related to these critical adaptations, e.g., adaptation to captivity.

20.     The February, 2014 and October, 2014 HGMPs do not adequately discuss or consider all of the results of the Johnson and Friesen study, or of other important studies. Instead, they focus on study results that suggest integration with wild fish could adequately maintain heterozygosity and allelic richness in hatchery Chinook populations. But there is very limited to no real consideration and discussion of the likely negative fitness effects of introgression from hatchery Chinook into wild Chinook. However, the February, 2014 HGMP does state the following: "brood stock integration may not fully mitigate negative effects of hatchery fish on the natural population. Chilcote et al. (2011) found no difference between integrated brood stock programs and segregated brood stock programs in terms of their relative impact on population intrinsic productivity, leading the authors to conclude that *integration may not be an effective means to eliminate the impact of hatchery programs on natural populations.*" (emphasis added).

21.     Dr. Johnson's rebuttal disclosures defend the use of a microsatellite assay and its interpretation in comparing McKenzie hatchery and wild salmon genetics. I agree that the microsatellite data are likely of reasonable quality. However, a relatively large number of missing loci per individual was allowed (Johnson and Friesen 2013), suggesting some poor quality data (individuals, and perhaps some loci) were apparently included. For example, the authors state: "we excluded all samples that amplified at fewer than 7 of 13 microsatellite loci from our analyses. Johnson and Friesen 2014; pg. 857 Methods section)." That said, the main problem is that the study uses few loci and uninformative (neutral or non-functional) loci compared to what is required and easily feasible (Hohenlohe et al. 2013, Narum et al. 2012). The few microsatellites used are apparently not mapped to chromosomes and thus it cannot be known how much of the genome or how many chromosomes have been assayed. Approximately 20,000 protein coding genes exist across the vast salmonid genome. The available data has extremely low power and low probability of detecting adaptive differences between hatchery and wild fish that would likely reduce fitness in the wild population following gene flow (introgression) from the hatchery.

22.     In his rebuttal disclosures, Dr. Johnson defends the use of four gene-linked satellite markers in establishing genetic similarity at important loci. Seventeen microsatellite markers, including just four that are gene-linked, reveal very little about adaptive differentiation. Dr. Johnson commits a fundamental error by characterizing a negative result in a low-power assay as significant evidence that no differences exist between the compared populations. Hundreds of mapped DNA marker loci on all chromosomes and evenly spaced would be more appropriate and sufficient. The results of the study Dr. Johnson refers to in his rebuttal disclosures show that the genetic distance between the wild and hatchery Chinook populations in the McKenzie River basin is less than the distance between them and other populations in the study, but these results say nothing about important genes under selection. Dr. Johnson fails to acknowledge in his rebuttal disclosures--in contrast to the acknowledgement in his 2013 study report--that his study does not (and cannot reliably) show whether adaptive genetic divergence has occurred in genes that govern reproductive fitness, which in turn influences viability of McKenzie Chinook

populations. If currently Dr. Johnson intends to infer that his study shows genetic identity in those important traits between the hatchery and wild populations, then he should clearly state so, and explain why statements to the contrary in his study report and in the reports of two independent panels are wrong.

**Levels of Introgression In the Context of Recovering Wild Fish Populations.**

23.     A 2009 Hatchery Scientific Review Group (HSRG) report states that the proportion of wild fish used in broodstock for integrated hatcheries with "primary" populations should exceed the proportion of hatchery fish that spawn among wild fish (the proportion of hatchery-origin spawners, or pHOS) by at least a factor of two, corresponding to a proportionate natural influence (PNI) value of 0.67 or greater. It also states that the maximum allowed pHOS depends on the percentage of natural origin broodstock (pNOB) in the hatchery broodstock, but in any case pHOS should never exceed 30%. In contrast, the proposed HGMP does not mention a PNI standard or goal for the McKenzie Hatchery program. Instead, it re-states the standard of < 10% pHOS, which was not listed by the HSRG as a standard recommended for integrated hatcheries, and whose origin is obscure and unexplained by documents I have reviewed in this case.

24.     In his rebuttal disclosures, Dr. Johnson defends divergence from HSRG guidelines. However, reflecting the HSRG requirements only "in many respects," to quote Dr. Johnson, is insufficient. If the HSRG hatchery best management practice guidelines, documented and published by the "independent scientific panel established and funded by Congress to provide an autonomous and credible evaluation of hatchery reform" (HSRG mission description; http://www.lltk.org/hrp-archive/HRP_HSRG.html) are ignored in a significant manner, it is incumbent upon the hatchery management agencies to provide an equally detailed scientific rationale for their actions.

25.     According to the McKenzie Hatchery Straying Technical Workgroup (*Assessment of Specific Actions to Reduce the Straying of Hatchery Chinook in the McKenzie River, McKenzie Hatchery Straying Technical Workgroup, January 27, 2012*), approximately 82% of the hatchery Chinook salmon that return to the McKenzie River are collected at the hatchery, with the remaining 18% staying in the river and available to spawn with wild salmon. This number is

estimated by assuming that stray hatchery salmon that remain in the river cross Leaburg Dam

fish ladders about 2 miles upstream of the hatchery, where they are counted, and ignores any

stray hatchery fish that do not cross the dam. Figure 3, ibid. However, large numbers of hatchery

salmon remain in the river below Leaburg Dam, and so would not be counted there. For instance,

in 2012, ODFW estimated that 542 of a total 821 hatchery-origin spawners in the McKenzie

River, or 66% of these spawners, were downstream of Leaburg Dam. Thus, the true proportion

of stray hatchery fish remaining in the McKenzie River, after collection of some of them at the

hatchery, can be assumed to be much higher than 18%.

26.     pHOS, which is the number of hatchery origin spawners divided by hatchery origin plus

natural origin spawners, is very often used as a proxy for introgression of hatchery genes into

wild fish populations. Measures to reduce potential genetic introgression usually focus on pHOS

as the relevant metric. The February, 2014 HGMP at table 2.2.2-4 states that the weighted

average pHOS for Chinook in the McKenzie River (excluding the population above Cougar

Dam) during the years 2005 through 2012 was 41%, with a pronounced upward (increasing)

trend over that period. Hatchery-bred Chinook in the McKenzie River basin return to spawn

between ages two and six years after they smolt and move into the ocean, with the largest

contingent returning at ages four and five. Therefore, the number of returning adult Chinook in a

particular year is a function primarily of the number of smolts released 4-5 years earlier. From

2001 through 2008, the average number of hatchery Chinook smolts released into the McKenzie

River basin was approximately 1,193,000. Given that 1,157,000 hatchery smolts were released in

2009, 1,180,000 in 2010, 1,223,000 in 2011, 1,199,000 in 2012, 882,000 in 2013, and 840,000 in

2014, and that ODFW now proposes to release 604,750 in 2015, it is likely that the pHOS of

Chinook in the McKenzie River is currently about the same as the recent average pHOS, and that

it will remain in or near this range for at least the next several years.

27.     In his rebuttal disclosures, Dr. Johnson asserts that pHOS is a poor proxy for gene flow

from hatchery to wild fish. I agree that there are better ways to measure genetic introgression. In

fact, that is one of the points of this declaration, and I make detailed recommendations on

feasible methodologies to improve the reliability of those measurements. Nevertheless, currently, pHOS is the accepted proxy the agencies use for introgression in the McKenzie River basin.

**Hatchery Fish Harm Wild Fish.**

28.     Hatchery domestication results from a process analogous to natural selection, but it occurs under unnatural conditions—the individual fish that are "selected" are those better adapted to live in unnatural conditions (high density, no predators, no disease or different disease, unnatural food, artificial spawning). The process results in loss of the ability to avoid predation, loss of disease resistance, and loss of ability to forage and spawn efficiently (Allendorf and Hard 2010). This artificial selection pressure is strong; it results in rapid adaptation to captivity with loss of the ability to survive and reproduce effectively in the wild (Allendorf et al. 2013). Genes (alleles) underlying these maladaptive traits will likely become fixed (or increase to high frequency) in hatcheries, even after only a few months of development and differential survival of embryos to smolts (Christie et al. 2014 and publications therein). These domestication effects occur even when all the hatchery fish are derived from the nearby local wild population and the hatchery operations regularly incorporate local wild fish into the hatchery broodstock.

29.     The McKenzie Hatchery Chinook stock is primarily but not completely derived from the local wild Chinook population, but it has had minimal and irregular incorporation of broodstock from the local wild salmon population. After many decades in the hatchery environment, it is extremely likely that hatchery Chinook from the McKenzie Hatchery have become highly domesticated.

30.     Maladaptive genes from hatchery Chinook will likely be transmitted to wild Chinook and reduce the fitness of wild Chinook if hatchery Chinook are allowed to spawn in the wild, as will occur when the returning hatchery Chinook stray onto the spawning grounds of wild Chinook. Such straying in the McKenzie River basin is documented, and is inevitable given the large number of smolts that have been released and are scheduled for release. The scientific literature clearly demonstrates that hatchery fish derived from local natural populations harm those same natural (or wild) populations (e.g., Araki et al., 2007a,b, Araki et al. 2009, Christie et al. 2011,

Christie et al. 2012, Christie, et al 2014, Normandeau et al. 2009; Theriault et al. 2011; Grant 2012). The most evident impact is reduced fitness. When hatchery fish are released into the habitat of wild fish, introgression of maladaptive genes into wild fish manifests in several ways. Reduced fitness is likely manifested as reduced survival of embryos, juveniles, and adults, which results from increased susceptibility to predation, disease, and stress in general (e.g. from pollution, climate warming, or variation in precipitation and stream flow associated with climate change or dams). Reduced survival likely also results, because fish with hatchery ancestry can have reduced competitiveness and the ability to acquire and retain quality habitat, shelter, food resources (fish compete for these things). Reduced fitness likely is also manifested as reduced reproductive success in adults, because they then show lower mating success, lower fecundity, and reduced viability of offspring. Introgression into wild Chinook from McKenzie Hatchery Chinook likely results in many hybrid (F1) individuals that fail to survive to reproduce, and thereby waste the genetic and reproductive resources of the wild parent.

31.     In his rebuttal disclosures, Dr. Johnson asserts that it is unreasonable to opine that the McKenzie Hatchery population contains maladaptive genes. Not only does that statement ignore the overwhelming and growing body of scientific evidence revealing negative effects of hatchery domestication, but in citing his 2013 study as evidence, Dr. Johnson ignores his own cautions in his 2013 report (as well as those of two independent panels, cited above) that his analysis was not relevant to adaptive or maladaptive genes; i.e., those that are under selective pressure. Dr. Johnson also notes that the HSRG found that hatchery programs "help preserve genetic resources in the ESU." Dr. Johnson fails to acknowledge that the HSRG clearly intended for hatchery populations to serve as a genetic resource only as a last resort, in those cases where the local wild population is extinct or nearly so. In fact, a major theme of the HSRG has been that in situations where a viable wild population exists (such as in the McKenzie), hatchery fish constitute a threat to its survival, not an aid (HSRG 2004, 2009).

32.     In his rebuttal disclosures, Dr. Johnson asserts there is no empirical evidence that maladaptive genes from [McKenzie] hatchery fish will likely be transmitted to wild fish and thereby reduce the fitness of the wild fish. If there is no empirical evidence specifically for the

McKenzie it is only because appropriate studies have not yet been conducted there. The overwhelming body of scientific evidence, from studies on Chinook salmon as well as other closely related salmonid species, shows that fitness-reducing domestication and genetic transmission occurs in situations similar to the McKenzie River. It would be preferable to have empirical proof of a local effect in hand, but in my professional experience it is not a prerequisite to concluding that such an effect is very likely occurring. And in fact, the steady decline of wild Chinook in the McKenzie River basin over the last decade is consistent with (and expected from) reproductive depression associated with the many millions of hatchery Chinook releases over that period.

33.     The HSRG categorizes hatchery stocks and associated hatchery programs as either "segregated" stocks (and programs) or as "integrated" stocks and programs, depending on the origins of the hatchery stocks, the manner in which the broodstock program is managed, and the intent of the hatchery program. A segregated program is one in which the hatchery stock is founded from a non-local population, receives no regular broodstock infusions from the local wild population, and is usually managed for the purpose of harvest. An integrated program is one in which the hatchery stock is founded from and regularly includes substantial additions of local wild fish as broodstock and may be intended to assist in the rebuilding of wild populations. In a 2004 report (Hatchery Scientific Review Group. April 2004. Principles and Recommendations of the Hatchery Scientific Review Group. p. B-4), the HSRG stated: "In practice, all hatchery programs must fall into one of the two categories; "intermediate" programs cannot exist without imposing significant risks to natural populations because of fundamental differences in the biological principles underlying the two types of programs."

34.     The HSRG has established guidelines for managing segregated programs due to concerns related to genetic introgression. These guidelines call for limiting the maximum pHOS to less than 5%. In my opinion, even this level of potential interbreeding between hatchery and wild salmon may be too high to limit or minimize negative fitness effects. The HSRG has also established guidelines for managing integrated programs. These guidelines attempt to minimize the genetic differences between the hatchery and wild populations by prescribing that a large

proportion of the hatchery broodstock will be replaced each year with wild, natural-origin broodstock; that the pNOB will be kept high; and that the pHOS of the naturally spawning population will be kept low, according to a sliding scale. In my opinion, even these difficult-to-meet provisions may be insufficient to limit or minimize negative fitness effects on the wild fish.

35.     The McKenzie Hatchery Chinook salmon program does not meet the HSRG definition of either a segregated or an integrated hatchery program, but rather appears to remain one of the "intermediate" programs that the HSRG says should not exist at all because they can have the most severely negative effects on wild fish fitness and recovery (Baskett and Waples 2013). The HGMP nevertheless claims that it is an integrated hatchery program, and sets as its modest management goals an average pNOB of 5-10% and a pHOS of < 10%. In my opinion, these standards are insufficient to limit or minimize negative fitness effects. Moreover, these standards have no generally accepted scientific foundation of which I am aware.

36.     In his rebuttal disclosures, Dr. Johnson noted that the HSRG's "Current" status of the McKenzie (circa 2009) concluded that the McKenzie River met the standard of primary populations for an integrated hatchery program. The HSRG listed in its report an effective pHOS in the river of 10%, and a PNI of 0.71. However, both of those values were derived from the management goals of a 2008 draft HGMP that the HSRG reviewed, rather than from existing verified data, and the values are wildly out of line with actual conditions on the river. To illustrate: (1) pHOS was considered only for the section of the river above Leaburg Dam; and even there, ODFW data shows that pHOS has been consistently much higher than 10%. More important, the relevant pHOS--for the entire McKenzie River basin--has averaged 40% or more over the last decade (see "Assessment of Alternatives" (2012)); and (2) the stated PNI assumes both a too-low pHOS of 10%, and a pNOB in the McKenzie Hatchery broodstock that is a way-too-high 25-40%. Data show that pNOB at the McKenzie Hatchery has never been close to those values. Moreover, given the low and declining number of wild salmon in the McKenzie, there is no realistic prospect of achieving a high pNOB in the foreseeable future. It is clear that the HSRG came to its 2009 -- but now inaccurate and inapplicable -- conclusion that the McKenzie

Hatchery met the definition of an integrated hatchery, because it based its conclusion on faulty numbers in a plan provided to it.

37.     In his rebuttal disclosures, Dr. Johnson refers to the HSRG's statement that "Options for improving hatchery programs...are limited due to the low number of natural-origin fish in the subbasin." This observation confirms my opinion. There is no reasonable chance of raising PNI to >0.67 by increasing pNOB, because there are too few wild Chinook spawners that can be mined to support this large hatchery program. Therefore, the only feasible approach to a higher PNI is to lower pHOS. And, the only sure way to lower pHOS is to reduce hatchery releases.

38.     Nonetheless, even given its shortcomings related to recovering wild spring Chinook salmon in the McKenzie River basin, the 10% pHOS standard is likely to be exceeded by a very large margin by the number of hatchery smolts that ODFW has released in recent years as those fish return and by the number ODFW proposes for release in the future.

39.     Given the many decades of domestication, a history that includes out-of-basin broodstock incorporation and low continuing incorporation of wild broodstock into the McKenzie Hatchery Chinook stock, the hatchery Chinook are expected to have a substantially reduced fitness and return rate compared to wild Chinook. McKenzie Hatchery Chinook spawning with wild Chinook would be expected to yield progeny with fitness intermediate between these. Consequently, interbreeding between wild and stray hatchery Chinook throughout the McKenzie River basin can be expected to reduce the fitness of wild Chinook that spawn with hatchery Chinook by an unknown but likely biologically significant amount (e.g., Christie et al. 2014). This imposes a substantial and likely severe genetically-based fitness burden on already depressed local populations of wild Chinook.  Some of the negative effects from hatchery gene introgression can persist for many years (generations) in the wild population, however few empirical data exist on duration of effects.

40.     The harm of hatchery introgression on wild fishes' ability to reproduce has been well documented in other salmonids, such as for coho salmon and steelhead trout. The findings of these studies apply equally to Chinook, because selection for adaptation to captivity has been strong in Chinook (like in other salmon and steelhead trout), and because empirical data from

Chinook also show that hatchery introgression reduces reproductive success in wild Chinook (Ford et al. 2013; Appendix of Hess et al. 2013; Christie et al. 2014; Banks et al. 2014). In fact, Christie et al. (2014) found a consistent reduction in reproductive success among "51 estimates from six studies on four salmonid species, showing that early-generation hatchery fish averaged only half the reproductive success of their wild-origin counterparts when spawning in the wild" and that "all species showed reduced fitness due to hatchery rearing" (emphases added). This reduced fitness was for hatchery fish created with local- and predominantly wild-origin parents, with little time (≤ 1 generation) in the hatchery.

41.     One study was of a wild winter-run steelhead supplementation program in the Hood River, Oregon, which was started in 1991 and continues. Conditions there allow the program to control the collection of adults for broodstock and the numbers of hatchery-produced steelhead that spawn naturally. It is important to note that such ideal conditions (bringing in new wild parents into the hatchery to reduce adaptation to captivity) are not present for most salmonid programs. The success of the Hood River program has been studied intensively by Dr. Michael Blouin and his colleagues at Oregon State University, with key results reported in numerous peer-reviewed scientific publications (including Araki et al. 2007a,b, Araki et al. 2009, Christie et al. 2011, and Christie et al. 2012). Araki et al. have shown that on average first-generation hatchery fish produced from native wild broodstock have only 60% to 80% of the reproductive success in the wild compared to the native wild fish themselves. Araki et al. have also shown that when second-generation supplementation hatchery fish (i.e., the offspring of wild-spawning first-generation hatchery fish) spawn in the wild, they also produce fewer surviving adult offspring than pure wild fish (W x W). The fact that the loss of fitness is a genetic effect (i.e., has a genetic basis), and was not an effect of the hatchery rearing environment, was demonstrated. It is likely hatchery Chinook introgression in the McKenzie River basin would cause even more severe reduction in fitness in wild fish than was observed in Hood River, because hatchery Chinook in the McKenzie River basin have not had large regular infusions (if any recent) of wild broodstock, and have been more strongly adapted to captivity for many generations (decades).

42.     In his rebuttal disclosures, Dr. Johnson states that the concept that McKenzie Hatchery salmon likely have lower reproductive success than wild fish is largely based on findings from other species. This is untrue. The effect has been observed in Chinook salmon as well as a variety of other closely related salmonid species. Observing fitness reduction associated with hatchery production has been a general phenomenon (almost a law now), given the large theory and massive empirical data accumulating among many taxa (all 5 salmon species and steelhead), particularly over the last decade. Contrary reports are rare, and considered outliers to the scientific mainstream or due to low statistical power (Christie et al. 2014, Box 2). See the example studies, which themselves constitute just a small sample of the large number of similar reports now in the scientific literature, which I list above. The effect is acknowledged as a general phenomenon of concern by virtually all fishery agencies, including NMFS and ODFW. The HSRG was commissioned by Congress more than a decade ago largely to implement hatchery reforms designed to avoid the negative effects of hatchery fish on wild fish. Indeed, all three of the references Dr. Johnson cites as examples of studies contradicting the general consensus fail to support the point he makes. All showed lower reproductive success for hatchery Chinook, but suggested in some cases a mechanism explaining the phenomenon. In one case, the effect was not shown to be statistically significant, but that was likely an outcome of the low statistical power of the study design (Christie et al. 2014). Thus, any attempt to dismiss the problem as minor, occurring only elsewhere, and only in other kinds of fish is disingenuous or uninformed at best.

43.     In his rebuttal disclosures, Dr. Johnson cites the preliminary data in an ongoing study in the South Fork McKenzie River (Banks et al., 2014) as evidence that hatchery Chinook do not have lower reproductive success in the McKenzie River basin than wild Chinook. However, Banks et al. (2014) does not compare hatchery fish to truly wild fish. In Banks's study, the naturally spawned wild parents in the study are recent progeny (first and second generation, etc.) of hatchery fish that were outplanted in the South Fork above Cougar Dam. So any fitness differences between hatchery and what we might call the semi-wild salmon are expected to be less than between hatchery and truly wild fish. Moreover, Reproductive Success (RS) as defined

in Banks' study is different than how RS is typically defined in other studies, making comparisons difficult. Nevertheless, the results to date are consistent with a lower reproductive success or total lifetime fitness for hatchery parents compared to semi-wild parents. The observations that Dr. Johnson describes about the size of male salmon <u>may</u> eventually provide an explanation of a <u>mechanism</u> behind the lower reproductive success, but they do not change the initial indications of the study that hatchery parents are, in fact, <u>less successful</u>. Dr. Johnson selectively focuses on data from the study that failed to show a difference specifically between <u>female</u> hatchery and semi-wild salmon, without pointing out that the statistical power of the study does not yet allow a conclusion that there is no real difference between them.

44.      In addition to the genetic impacts from the interbreeding of hatchery Chinook with wild Chinook in the wild in the McKenzie River basin, there is a separate demographic effect on the number of wild spawners that occurs, due to the lower fitness of wild fish that interbreed with hatchery instead of with other members of the wild population: each generation, fewer pure wild adult fish return because fewer wild fish bred with one another than in the previous generation. If any of the progeny of the interbreeding of hatchery and wild Chinook do survive to return, the total returning adult population will still be smaller than if only wild fish bred with one another because each hatchery-wild mating will produce fewer returning adults than a wild-wild mating would have. If wild-hatchery matings produce no returning adults the returning adult population will be made up of only the offspring of wild-wild matings, but the total, purely wild, population will be lower in number still. Consequently, even in the extreme case where wild-hatchery matings result in no gene flow from hatchery spawners to the local wild population, the lower fitness of wild-hatchery matings in the preceding generation still produces a serious negative, though purely demographic, impact on local wild populations. This effect constitutes a real and significantly likely threat to the viability of wild Chinook in the McKenzie River basin, along with existing and emerging disease threats (Roberts 2012; Krkosek et al. 2011). Hatcheries hold fish at high densities which increases risk of high disease prevalence and transmission. The negative demographic impacts of hatchery and disease risks in the McKenzie River basin, given

the importance of wild Chinook in the McKenzie River, also threaten the viability of the entire Upper Willamette River ESU.

45.     Introgression from hatchery Chinook released from the McKenzie Hatchery into wild Chinook in the McKenzie River basin is very likely to result in hybridized individuals that either fail to survive to reproduce—thereby wasting the demographic and genetic resources of the wild parent—or they produce offspring whose reproductive fitness is lower than wild fish, resulting in offspring that contribute to a gradual lowering of the average fitness of the wild population. This type of introgression might not manifest itself as a hybrid swarm (at least initially) whereby most or all individuals in the wild/hybrid/hatchery population contains a mixture of genetic material from the wild and hatchery populations. Rather, introgression will likely initially result in a small to moderate number of individuals with a proportion of hatchery genes accompanied by the gradual depression of the fitness of the surviving members of the wild population.

46.     Detecting and quantifying the extent of introgression will depend on several factors, including the frequency of cross-breeding, the manner by which genetic samples from the wild population are obtained, the type and genome position of genetic markers employed, and the number of genetic marker loci employed. To detect introgression, it is now standard procedure to genotype 100 to 1000 population-diagnostic or informative markers (Hohenlohe et al. 2013 and 2013; Amish et al. 2012; Hand et al. in press). Tests for introgression among hatchery-bred and wild Chinook in the McKenzie River basin should include many tens of population-informative markers, rather than a dozen markers of relatively low population-diagnostic informativeness (e.g., low genetic differentiation, i.e., $F$st), such as the microsatellite DNA markers that have been used. Use of many tens of such (informative) DNA markers is the only way to ensure detection and reliable quantification of admixture, especially in cases where only a small proportion of the genome from the maladapted population (e.g., hatchery fish) is likely to introgress (Hohenlohe et al. 2013).

47.     The risks posed by the hatchery program are exacerbated by the lack of informative genetic markers but also the lack of sufficient monitoring for introgression of hatchery genes into wild fish populations (e.g., maladaptive genes that are driven to fixation or high frequency in

hatcheries). It is essentially unknown how much harmful introgression has occurred whether or not the hatchery programs will be properly monitored, nor whether sufficient funding exists for timely monitoring and evaluation of the likely harmful effects of hatchery actions. Rigorous genetic monitoring and evaluation of hatchery programs and of their impact on wild salmonids are essential to preventing unwarranted and severe harm to wild fish populations (Schwartz et al. 2007). Improved and continual monitoring is especially important given the rapidly improving ability of biologists to use molecular and computational tools to monitor gene flow and the effects of hatchery introgression on individual fitness and population performance (*i.e.*, on population growth rate and the probability of population persistence).

48.     The lack of data and plans for measuring the rates at which returning hatchery-bred adults reproduce in the wild and for comparing the reproductive success and survival rates of hatchery versus wild Chinook, poses additional concerns. Unless the relative reproductive success of the hatchery fish can be monitored in an accurate and timely manner, the annual growth rate of the wild population could be declining while the numerical increase in the total population spawning in the wild annually is stable or increasing due to the returns of or introgression from hatchery fish. It is critical for the program to have standards in place to limit these harmful effects and monitoring in place to detect them in a timely fashion.

49.     A particular concern with introgression among hatchery and wild Chinook in the McKenzie River basin is the likelihood that a large proportion of progeny from matings between them may die at young juvenile ages before they can be included in genetic samples. This is problematic because these negative (early life stage) effects of hatchery introgression would not be detected (unless embryos and age 0 fish are sampled). This would produce another deleterious demographic impact on wild populations distinct from genetic impacts; however, loss of progeny of hatchery-wild matings at young juvenile ages will considerably complicate the detection of this interbreeding by genetic methods. It is very important that a sampling study design aimed at detecting the genetic signals of interbreeding between McKenzie Hatchery and wild Chinook include acquiring samples of fertilized eggs deposited in spawning nests (redds), or at least of juveniles during the first month or two following emergence of fry from the gravel. Reliable

detection of cross-breeding and assessment of negative effects of introgression requires sampling a few embryos from each of many tens or 100s of redds and/or fry and genotyping with many tens or 100s of DNA markers.

50. Even low rates of interbreeding in the wild between hatchery and wild Chinook in the McKenzie River basin will likely have a harmful impact on the wild Chinook population, and on its long-term fitness and adaptive capacity, i.e., its ability to survive, breed, and produce viable offspring in future environments. Such impacts will further threaten the viability and potential for recovery of wild Chinook in the Upper Willamette River ESU. The ESU would best be protected from this threat by terminating releases of hatchery Chinook into the McKenzie River basin. Interbreeding of hatchery with wild Chinook will likely harm the wild population and poses a threat to the viability and adaptive potential of that population. Short of terminating releases of hatchery-bred fish, reducing their threat to a low level is essential and will require significantly reducing the number of hatchery-bred smolts that are released.

51. In his rebuttal disclosures, Dr. Johnson disputes that release of lower numbers of smolts (particularly my recommendation above, of no more than 77,000 smolts) is appropriate. I think that far less than the planned 604,750 smolts should be released annually, because the fraction of those releases that return as adults will cause significant harm to wild Chinook and slow or prevent the recovery of wild Chinook populations. I think that zero, or at most 77,000 hatchery smolts should be released, which would allow for approximately 400 returning adults per year, assuming a smolt-to-adult-return ratio of about 0.59%. This is based on my understanding of the current best available science and my assessment that, in the McKenzie River basin, straying of hatchery Chinook and subsequent interbreeding with wild Chinook presents a high risk of reducing reproductive fitness.

52. Recent introgression, as well as introgression that occurred further in the past as a result of very large hatchery smolt releases for many decades, (approximately 24 million just during the period 1978-2008 (Johnson and Friesen (2010) table 3.8) would have lowered the productivity of the wild population. Thus, the current productive capacity of the wild Chinook is in part very likely a legacy of the past decades of hatchery smolt plants, among other

contributing factors. This impaired productivity makes it even more critical that opportunities for introgression of McKenzie Hatchery Chinook genes into the wild Chinook population be eliminated or greatly reduced in order to maximize the opportunity for the wild salmon population to begin to recover its productive capacity in the context of current and future environmental selective regimes (natural selection in the wild).

53.     Releasing 604,750 hatchery smolts into the McKenzie River basin will very likely cause significant harmful effects to wild Chinook in the McKenzie River basin. These harmful effects include reduced abundance of wild adult salmon, due to the failure of the offspring of many of the hatchery-wild matings to survive to adulthood. They also include lowered reproductive performance of the progeny of hatchery-wild matings that did survive to return to reproduce in the wild. The latter would have resulted in the introgression of hatchery genes into the wild population. These individuals, progeny of hatchery-wild matings, would not have missing adipose fins like their hatchery parents, but rather would appear to be wild fish (indeed, they would be "natural-origin" fish). Some of these individuals would likely have been included in adult samples used to estimate the genetic composition of wild Chinook, and this would increase the odds that introgressed individuals would be very difficult to distinguish from true wild adults by the recent genetic tests described in the Johnson and Friesen study.
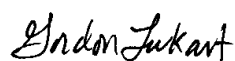
54.     Another harmful impact of hatchery Chinook releases into wild Chinook runs is that supplementation often reduces the genetically effective population size of the overall population. This is called the Ryman–Laikre effect, whereby hatchery fish supplementation leads to a reduced overall effective population size ($N_e$) due to increased variance in reproductive success among individuals, e.g., relatively few hatchery adults produce many offspring (Ryman and Laikre 1991). This reduced $N_e$ increases the rate of loss of genetic variation over time (in the hatchery and the overall hatchery-wild population) and also reduces the efficiency of natural selection for local adaptation, even if the census size of the population increases from hatchery supplementation. As one example, Christie et al. (2012) found that, in the wild, "the addition of hatchery fish doubled the total number of adult fish on the spawning grounds each year, but cut the effective population size of the total population (wild and hatchery fish combined) by nearly

two-thirds." The only way to know the effective size of the wild population and to know if hatchery supplementation reduces the $N_e$, is to use genetic markers to estimate and monitor the $N_e$ (e.g., Luikart et al. 2010; Waples et al. 2014). Thus, it is important that the effective population size of the wild populations (e.g., in the mainstem McKenzie, and above Cougar Dam) and also the hatchery population be estimated and monitored using genetic markers (e.g., Schwartz et al. 2007). Unfortunately, the set of genetic markers in the HGMP (10-20 microsatellites), and statistical approaches are insufficient (and insufficiently explained) to ensure reliable early detection of the negative effects of supplementation on the wild and overall effective populations size of spring Chinook salmon in the basin.

55.     The McKenzie Hatchery Chinook program significantly harms wild Chinook in the basin, and the ability of the wild Chinook population to recover. Hatchery-bred salmon (and their offspring) very likely have substantially reduced fitness compared to wild salmon due to decades of domestication and strong selection causing genetic adaptation to a hatchery environment (Banks et al. 2014; Christie et al. 2014). Some adult hatchery-bred salmon likely cross-breed with wild salmon, and it is highly likely that the resultant offspring have reduced fitness (i.e., survival and reproduction) in the wild, especially when considering the many millions of hatchery smolts released over decades. Some maladapted genes introduced into the wild Chinook population will likely persist for many generations even after the returns of hatchery fish have ceased, thereby causing long-lasting harm to the populations. This harm from hatchery introgression, along with the negative demographic effects, increased disease threats and a potential reduction of the effective population size is especially problematic because the McKenzie River spring Chinook population is a "genetic legacy" population, critical to recovery of the entire Upper Willamette River Basin ESU.

I declare under the penalty of perjury that the foregoing is true and correct.

Date: December 4, 2014.

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1987, Research and Teaching Assistant, Sumilon University, Philippines (SCUBA diving & Marine Biology)

1986, Field Research Assistant, Virginia Polytech Institute (trapping & banding passerine birds)

1985-1986, Iowa Department of Natural Resources (gill-netting, radio-telemetry of fish, grouse, & otters)


ACADEMIC HONORS:

Adjunct Professor (can supervise grad students), Wildlife Program, University of Montana, 2010-current.

Bronze medal, a top scientist in France CNRS (Centre Nationale de la Recherche Scientifique), 2004-2005.

Doctoral Research Fellowship, University of Montana, 1996.

Fulbright Fellow, La Trobe Univ., Melbourne, Australia, 1994-95 (Genetics of Endangered Marsupials).

Scholarship: top 5% of graduate students in research at The University of Montana, 1991, 1994.

Phi Beta Kappa National Honors Society Award (top 5% of liberal arts students in USA), 1988

PROFESSIONAL ACTIVITIES:

2001-2004, Journal editorial board member for Conservation Biology

2003-2006, Journal associate editor for Molecular Ecology Resources

2009-2011, Associate editor for Journal of Heredity

2001-current, Member IUNC Caprinae Specialist Group

2006-2011, ConGen International Course on Population Genetic Data      Analysis (http://popgen.eu/congen2011/)

2010-curent, Member Swan Ecosystem Center Native Fish Committee


SOCIETIES (Last five years):

American Fisheries Society

Ecological Society of America

Society for Conservation Biology

Society for the Study of Evolution

Wildlife Disease Association

Wildlife Society


BOOKS:

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For some see: http://www.ncbi.nlm.nih.gov/sites/entrez?cmd=search&term=Luikart%20G

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Hogg, J.T., S.H. Forbes, B.M. Steele, and G. Luikart. 2006. Genetic rescue of an insular population of large mammals. Proceedings of the Royal Society, 273:1491-1499. Jordan, S., C. Miquel, P. Taberlet, and G. Luikart. 2006. Sequencing primers and SNPs for five rapidly evolving reproductive loci in endangered ibex and their kin (Bovidae, Capra spp.), Molecular Ecology Notes, 6:776-779.

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Antao, T., I.M. Hastings, G. Luikart, M.J. Donnelly. Estimating effective population size in disease vectors: a critical assessment of applications and performance. In review.

Antao, T., G. Luikart et al. Detecting $F_{ST}$ -outliers and directional selection requires genotyping multiple SNPs per gene: lessons from empirical genomic data. In prep.

Kardos, M., G. Luikart, F.W. Allendorf. Evaluating the role of inbreeding depression in heterozygosity-fitness correlation: how useful are tests of identity disequilibrium? In review.

Cosart, T., A. Beja-Pereira, J. Johnson, and G. Luikart. ExonSampler: A computer program for genome-wide sequence sampling to facilitate new generation sequencing. In prep.

Almendra, C. et al. Detecting brucellosis in wildlife: consequences for public health and disease eradication. In review.

Antao, T., A. Pérez-Figueroa, I.M. Hastings, M.J. Donnelly, and G. Luikart. Interpreting estimates of effective population size and heterozygosity: caveat emptor! In review.

Amish, S., Y. Hoareau, C. Almendra, N. Anderson, P.R. Clark, H. Edwards, R. Frey, M. Gruber J. Henningsen, R. Wallen, G. Luikart. Sensitive pathogen detection in nonlethal and noninvasive ungulate samples using PCR. In prep.


SELECTED GRANTS AND CONTRACTS AWARDED OR CONTINUED (Selected recent grants & contracts):

Grants Awarded or Continued as Principal Investigator/Project Director:

NSF–EID (Ecology of infectious diseases): Effects of land-use and predation risk on wildlife contact networks and Brucella transmission in the Yellowstone Ecosystem. 2010-2014.

NSF-EID: Microparasite-Macroparasite Interactions: Dynamics of Co-infection and Implications for Disease Control. V. Ezenwa, A. Jolles, G. Luikart, E. Nunn. 2007-2011.

NSF-IGERT: Montana Ecology of Infectious Diseases: Integrative Graduate Training on Multi-scalar Computational, Mathematical and Empirical Approaches to Complex Biological Problems. Added as co-PI with Bill Holben, Jesse Johnson, Jonathan Graham. 2006-2012.

NSF-OPUS: Evolutionary genetics and the conservation of exploited populations. DEB0639770. Co- PI Gordon Luikart with Fred Allendorf (PI). 2008-2011.

USGS-PNW Climate center: Predicting Climate Change Impacts on River Ecosystems and Salmonids across the Pacific Northwest: Combining Vulnerability Modeling, Landscape Genomics, and Economic Evaluations for Conservation. Funded 2012-2014. $208,000.

ARC (Australian Research Council) Linkage grant funding for a research project entitled "Genomics for persistence of Australia freshwater fish". P. Sunnucks et al. 2010-2015.

USFWS: Development and application of SNPs for estimating the number of breeders in lake trout following suppression. 2012-2013.

MFWP: New DNA Markers to assess hybridization, local adaptation, and restoration success in bull trout. 2011-2014.

MFWP: Hundreds of new SNP markers to detect hybridization in westslope cutthroat trout. 2011-2014.

MFWP: Development and Validation of Q-PCR Tests for Early Detection of Dreissenid mussels. 2011-2012.

USFS: Admixture and diversity assessments in westslope cutthroat trout of the Swan River drainage: SNP-chip analyses, 2011-2013.

POBS (Park Oriented Biological Research): Developing non-invasive techniques for bighorn sheep population estimation using fecal DNA. Kathryn A. Schoenecker, et al. 2009-2010.